Resection and Graft Replacement of an Aneurysm of the Ascending Thoracic Aorta With Simultaneous Repair of Aortic Valvular Insufficiency

PETER G. GAAL, M.D., and
JAMES V. MALONEY, JR., M.D., Los Angeles

AN ANEURYSM of the ascending thoracic aorta frequently is accompanied or is followed by aortic insufficiency. It may arise from dilatation of the annulus of the aortic valve by expansion of the adjacent arterial wall, but it may also be the end result of a disease process that has affected both arterial wall and valve structure.

Aortic insufficiency and aneurysm of the ascending aorta have been dealt with as separate entities by a number of investigators. Muller and associates presented a comprehensive review of the various surgical approaches currently employed for the treatment of aortic insufficiency.[6] Bahnson and Spencer reported their results in eight cases of aneurysm of the ascending aorta.[1]

Although most aneurysms of the ascending aorta are the result of atherosclerosis or syphilis, a significant number arise as the result of cystic medial necrosis of the aortic wall. In 1930, Erdman described "mediconecrosis aortae idiopathica cystica," in which morphologic findings were similar to those noted in the vessels of patients with Marfan's syndrome.[4] (This relationship was described by Baer, Taussig, and Oppenheimer in 1942.)[2] Weaver, Edwards and Brandenburg postulated that idiopathic dilatation of the aorta with aortic valvular insufficiency may be a *forme fruste* of Marfan's syndrome.[7]

Ellis, Cooley, and DeBakey coined the term *annulo-aortic ectasis* to describe the coexistence of aortic insufficiency and an aneurysm of the ascending aorta.[3] In the case they reported, end-to-end anastomosis of the divided aorta was accomplished without the necessity of inserting a graft. The following case report is presented to illustrate the manner in which principles of heart and blood vessel surgery may be applied to these problems.

From the Department of Surgery, University of California Medical Center, Los Angeles 90024.

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REPORT OF A CASE

The patient, a 42-year-old white man, had been employed as a truck driver but was forced to stop work in early 1960 because of increasing angina and episodes of tachycardia associated with lightheadedness and transitory blindness. He first began to notice exertional dyspnea and fatigue in 1952, and at that time an aortic diastolic murmur was detected. The symptoms gradually progressed until the patient was put in hospital in December, 1960, for treatment of congestive failure. Following administration of digitalis and diuretics, he improved sufficiently to return home, although he was unable to resume his former employment.

In July, 1961, the patient was admitted to the University of California Medical Center, Los Angeles, with complaint of increasing disability due to chest pain and shortness of breath. He recalled having had several attacks of tonsillitis in childhood but was unaware of any long illness. At age 22, he had had rheumatic joint pains which subsided gradually over a period of several weeks. In 1943, he was put in hospital for pulmonary tuberculosis involving the right upper lobe. In 1958 resection of the apical segment of the right upper lobe was carried out.

Upon physicial examination the patient was observed to be thin and he appeared chronically ill. When he sat erect, some bobbing of the head occurred. Blood pressure ranged from 145/20 to 170/50 mm of mercury. The pulse rate was 80 per minute, with an occasional irregular beat. The precordial region was hyperactive, and a prominent left ventricular heave was noted. A thrill was present over the proximal aorta and at the apex of the heart, and a strong aortic thrust over the right side of the sternum. An early Grade III diastolic murmur was present at the aortic area, and a late Grade II systolic murmur in the same region. The peripheral pulses were accentuated, and capillary pulsations notable in the nail beds. Duroziez's murmur was present over the femoral arteries.

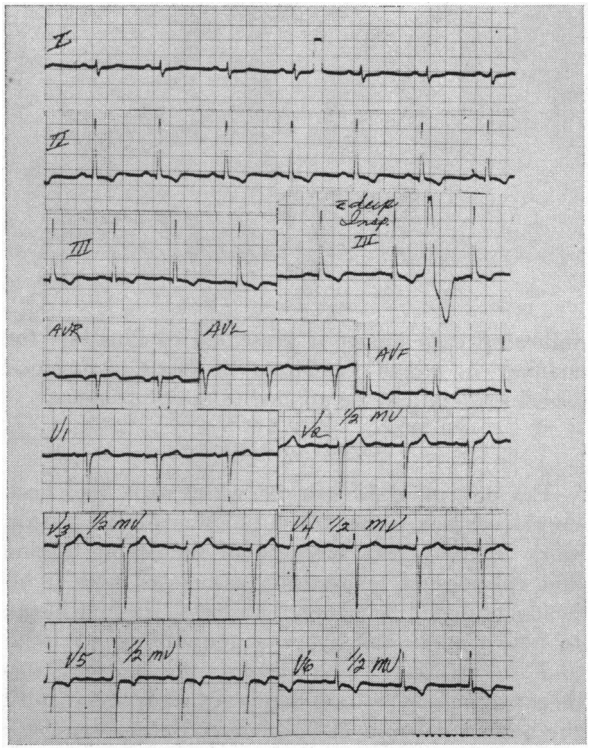

Figure 1.—Preoperative electrocardiogram consistent with biventricular hypertrophy.

The electrocardiogram was interpreted as showing biventricular hypertrophy, with changes more prominent over the left ventricle (Figure 1). X-ray films of the chest showed moderate cardiac enlargement. The ascending aorta was thought to be dilated, and calcification was noted in the proximal aorta. The arch was prominent (Figure 2).

The chest was opened by median sternotomy and an aneurysm was noted along the right anterolateral surface of the aortic wall. The aneurysm was saccular in configuration, and thin plates of calcium were palpable within its wall. The left ventricle was greatly enlarged. Upon cannulating the femoral artery, advanced atheromatous changes were noted, which lent support to the impression that a similar process was at least partly responsible for weakness of the aortic wall.

The ascending aorta was mobilized from the level of the right coronary artery to the innominate artery. Extracorporeal circulation was begun, and body temperature was lowered from 37°C to 31.5°C. The aorta was cross-clamped proximal to the innominate artery, and the anterior wall of ascending aorta was incised to gain exposure of the aortic valve (Figure 3A). The usual tricuspid configuration was found. However, the annulus appeared dilated to approximately twice normal, and the cusps somewhat thickened, foreshortened and stiff. The inner wall of aorta was severely involved with atheromatous change, hard in some areas, thin and fragile in others. There was no evidence of dissection from the adjacent aneurysm. The coronary arteries were cannulated (not shown in diagrams), and oxygenated blood was delivered to the heart throughout the procedure. Blood entering the left atrium was recovered by inserting a

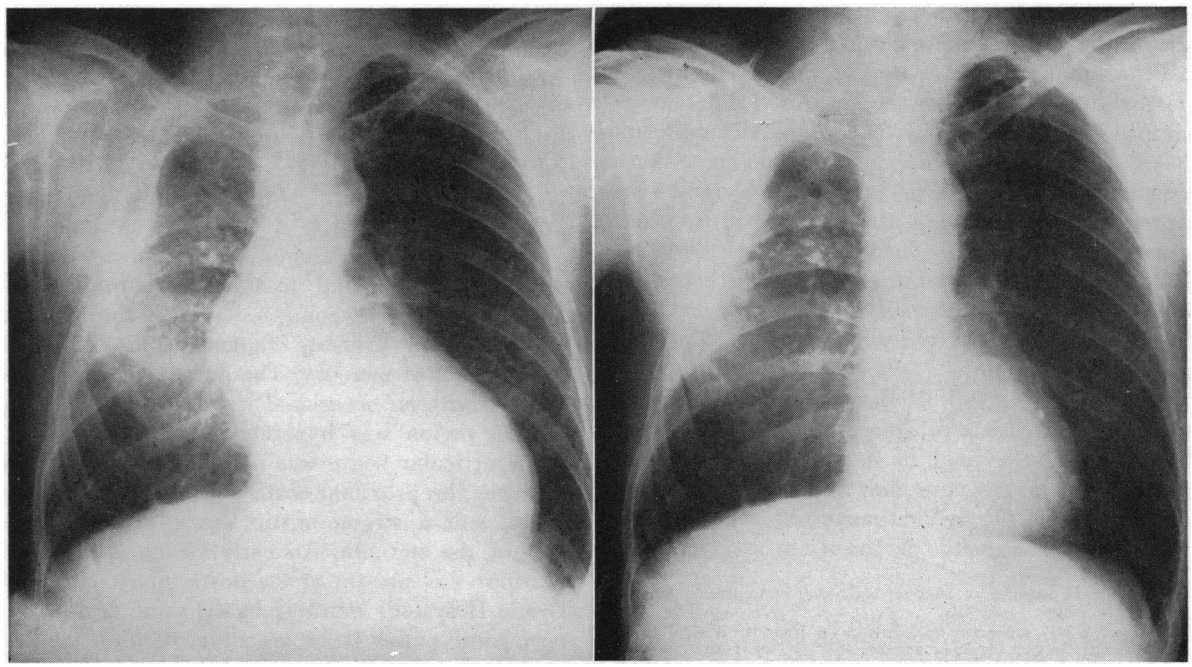

Figure 2.—Preoperative and postoperative x-ray films of the chest. The pulmonary changes are the result of old tuberculosis.

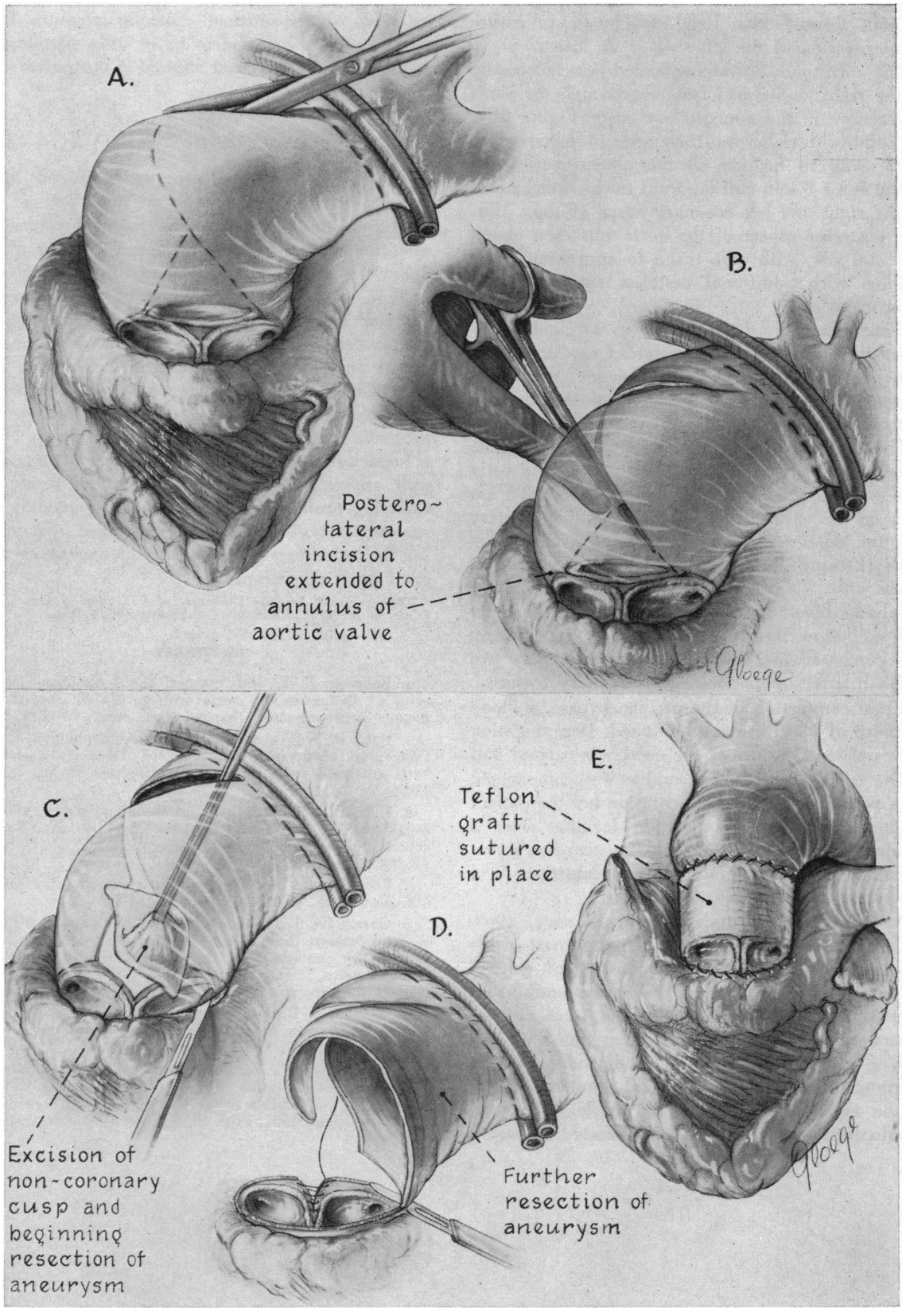

Figure 3.—*A, B, C,* excision of noncoronary leaflet to produce a bicuspid aortic valve; *D, E,* resection of involved segment of aorta and insertion of graft.

cannula through the atrial appendage to insure decompression of the left side of the heart.

The aortic incision was extended posterolaterally to the right in a spiral fashion, through the sinus of Valsalva of the noncoronary cusp (Figure 3B). An elliptical incision was then made in the posterior aortic wall to include all the noncoronary cusp except for a 3 mm cuff adjacent to the attachments of the right and left coronary cusps (Figure 3C). The posterior aspect of the aorta was then closed with 2-0 silk, with care taken to approximate the annulus with additional mattress sutures of the same material.

Bicuspidization of the valve, as originally proposed by Garamella and associates,[5] produced complete correction of the insufficiency previously present without significantly compromising the size of the orifice.

The segment of aorta between the innominate artery and a point just distal to the coronary ostia was excised, leaving a cuff of aorta about 2.5 cm long on the cardiac side of the innominate artery (Figure 3D). A crimped, closely-woven Teflon graft (Edwards type) 3.5 cm long was sutured in place with 3-0 silk (Figure 3E). The cannulas supplying blood to the coronary vessels were removed before insertion of the last few stitches. The combined procedures took approximately two and a quarter hours of cardiopulmonary by-pass.

Upon completion of the cardiac bypass, a blood pressure of 140/80 was maintained. Decannulation of vessels and closure of the chest was carried out.

The early postoperative course was complicated by a period of severe disorientation but the patient gradually recovered his mental faculties over a ten-day period. Throughout this time, cardiac action remained good and blood pressure continued in a normotensive range.

At a follow-up examination in January, 1962, the patient was found to be much improved in his tolerance to exercise and his feeling of general wellbeing. He continued to have some mild subjective neurologic symptoms which were not incapacitating. The blood pressure at this time was 150/88 mm of mercury. A low-intensity systolic murmur was heard in the second intercostal space at the right sternal border. A Grade I-II decrescendo diastolic murmur was also detected. However, in view of the normal diastolic pressure these murmurs were believed to be of little significance. X-ray films of the chest showed a normal cardiac silhouette (Figure 2).

DISCUSSION

The development of progressive aortic insufficiency in the presence of an aneurysm of the ascending thoracic aorta has received increasing attention during the past few years because of the difficulties encountered in attempting surgical correction. The inherent problems of treating aortic valvular incompetence are further complicated by the necessity of effectively dealing with the adjacent aneurysm, since both lesions threaten life. Perfusion of the coronary system during the period of aortic cross-clamping, the use of closely-woven synthetic fabrics which will not leak while the blood is heparinized, and meticulous suturing of the aortic wall are essential features of surgical treatment. Complex problems such as those presented in the case here reported can thus be managed by combining the techniques acquired through experience with open heart operations.

Department of Surgery, University of California at Los Angeles School of Medicine, Los Angeles, California 90024 (Gaal).

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